

REMARKS

I. Amendments

Claims 1-10 and 17-24 are canceled, and new claims 39-58 are added. The newly added claims do not introduce new matter and are completely supported throughout the application as originally filed. More particularly, newly added claims 39-43 directed to targeting constructs, methods of producing the targeting constructs, and transformed cells are supported, for example, at page 13, line 33 through page 21, line 28, and at page 57, line 8 through page 61, line 22 of the specification. Support for newly added claims 44-56 directed to a transgenic mouse comprising a homozygous disruption in a lymphoid-specific GPCR gene and a method of producing said transgenic mouse, and to cells and tissues isolated from the transgenic mouse may be found, for example, at page 21, line 29 through page 23, line 24, or at page 61, line 24 through page 62, line 27 of the specification. Lastly, new claims 57-58 directed to a transgenic mouse comprising a heterozygous disruption in a lymphoid-specific GPCR gene, and cells and tissues isolated from the transgenic mouse are supported, for example, at page 23, lines 13-18 of the specification.

The foregoing amendments are made solely to expedite prosecution of the application and are not intended to limit the scope of the invention. Moreover, the amendments to the claims are made without prejudice to the pending or now canceled claims or to any subject matter pursued in a related application. Applicants reserve the right to prosecute any canceled subject matter at a later time or in a later filed divisional, continuation, or continuation-in-part application.

Upon entry of the amendment, claims 39-58 are pending in the instant application.

II. Rejections

A. Rejection under 35 U.S.C. § 112, first paragraph

Claim 8 was rejected under 35 U.S.C. § 112, first paragraph, because the specification, while being enabling for a lymphoid-specific GPCR gene knockout mouse exhibiting the phenotype of lymphocyte infiltration, does not reasonably provide enablement for any other lymphoid-specific GPCR gene knockout non-human animals without said phenotype. Applicants respectfully traverse this rejection. In view of the cancellation of claim 8, the Examiner's rejection under 35 U.S.C. § 112, first paragraph, is no longer relevant.

Claims 17-23 were rejected under 35 U.S.C. § 112, first paragraph, on the grounds that the specification, while being enabling for a homozygous lymphoid-specific GPCR knockout mouse which lacks production of functional GPCR protein, does not reasonably provide

enablement for a heterozygous lymphoid-specific GPCR knockout mouse or a lymphoid-specific GPCR gene disrupted mouse. The Examiner asserts that the specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims. Applicants respectfully traverse this rejection. However, in light of the cancellation of claims 17-23, the rejection under 35 U.S.C. § 112, first paragraph, is moot.

Applicants submit that new claims 39-58 are fully enabled by the teachings of the specification. As the rejections under 35 U.S.C. § 112, first paragraph, of claims 8 and 17-23 are no longer relevant as a result of the cancellation of these claims, and new claims 39-58 are fully enabled by the teachings of the specification, Applicants respectfully request withdrawal of the rejection under 35 U.S.C. § 112, first paragraph.

B. Rejection under 35 U.S.C. § 112, second paragraph

Claims 1-4, 9, 10, 17, 18, and 20-24 were rejected under 35 U.S.C. § 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention. In particular, claims 1-4 and 10 were rejected under 35 U.S.C. § 112, second paragraph, on the basis that the arrangement of the targeting construct is unclear. Claims 9 and 24 were rejected under 35 U.S.C. § 112, second paragraph, on the basis that the word “derived” renders the claim indefinite because the nature and number of derivative processes is unknown. Claims 17, 18, and 20-24 were rejected under 35 U.S.C. § 112, second paragraph, on the grounds that the term “cellular infiltration” renders the claims indefinite because the nature of the cell and the location the cell infiltrates are unknown. Applicants respectfully traverse each rejection under 35 U.S.C. § 112, second paragraph. However, in light of the cancellation of claims 1-4, 9, 10, 17, 18, and 20-24, these rejections under 35 U.S.C. § 112, second paragraph, are moot. Applicants respectfully request withdrawal of the rejection under 35 U.S.C. § 112, second paragraph.

Applicants submit that new claims 39-58 are definite and particularly point out and distinctly claim the subject matter regarded as the invention in accordance with 35 U.S.C. § 112, second paragraph.

C. Rejections under 35 U.S.C. § 103

Claims 1-8 and 10 were rejected under 35 U.S.C. § 103 (a) as being unpatentable over Mansour *et al.*, 1988, *Nature*, 336(24):348-352 ("Mansour"), in view of Schweickart *et al.*, 1994, *Genomics*, 23: 643-650 ("Schweickart").

Applicants submit that new claims 39-58 are non-obvious over the teachings of the references cited. The claimed invention relates to the *in vivo* mammalian characterization of lymphoid-specific GPCR genes and methods and compositions relating thereto, all of which are not obvious in view of the sole or combined teachings and disclosures of Mansour and Schweickart.

According to the Examiner, Mansour teaches a strategy for targeted disruption of the *hprt* and proto-oncogene *int-2* in mice embryonic stem cells, and subsequent generation of knockout mice. The disclosure of Mansour specifically relates to a general method for isolating embryonic stem cells containing a targeted mutation in an endogenous gene. More particularly, Mansour teaches the targeted disruption of the *hprt* gene and the proto-oncogene *int-2* in mouse embryonic stem cells by homologous recombination using targeting constructs specific for these genes.

Schweickart, as characterized by the Examiner, teaches the cloning of the human and mouse lymphoid-specific GPCR gene designated EB11 (for Epstein-Barr induced 1), and provides the cloned coding sequence for this gene. Further, the Examiner asserts that Schweickart teaches that EB11 is highly homologous to several members of the leukocyte chemotactic peptide receptor family and that its expression is specific to lymphoid organs. Further, according to the Examiner, Schweickart teaches that EB11 may play a role in lymphocyte growth, differentiation, activation, leukocyte trafficking, and in the extravasation of blood cells into sites of inflammation.

As described above, the disclosure of Mansour is limited to providing a general approach for isolating embryonic stem cells. As acknowledged by the Examiner, Mansour provides no disclosure or teaching of how to make a lymphoid-specific GPCR gene targeting construct or lymphoid-specific GPCR gene knockout mouse (See Office Action, page 8). More particularly, Mansour does not teach or suggest a targeting construct containing a DNA sequence homologous to a lymphoid-specific GPCR gene or methods of producing such a construct as recited in the pending claims. Nor, does Mansour teach a method of producing a transgenic mouse comprising a homozygous disruption in a lymphoid-specific GPCR gene as claimed by the present invention.

Similarly, Schweickart does not teach or suggest a disruption in a lymphoid-specific GPCR gene. In particular, the disclosure of Schweickart fails to provide any teaching or suggestion that relates to the use of targeting constructs for creating a disruption in a lymphoid-specific GPCR gene, methods of producing such targeting constructs, and cells transformed with such a targeting construct, as recited in the pending claims. Schweickart further fails to provide any teaching or suggestion relating to transgenic animals, and, in particular, transgenic mice, comprising a disruption in a lymphoid-specific GPCR gene, or to cells, tissues, and methods relating thereto, as recited in the pending claims.

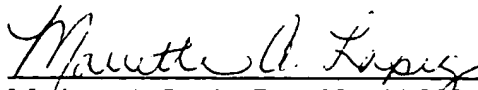
Taken together, the disclosures of Mansour and Schweickart are absent of any teaching or suggestion to disrupt the lymphoid-specific GPCR gene, and in particular, do not teach or suggest the transgenic mice, targeting constructs, tissues, cells, and methods as claimed in the present invention. More particularly, the disclosures of Mansour and Schweickart, alone or in combination, do not teach or suggest in any way transgenic mice comprising a disrupted lymphoid-specific GPCR gene as currently claimed by the present invention. Nor do Mansour and Schweickart, alone or in combination, teach or suggest a targeting construct, tissues or cells that are related to disruptions in lymphoid-specific GPCR genes as claimed by the present invention.

As the obviousness rejection is no longer relevant as a result of the cancellation of claims 1-8 and 10, and as new claims 39-58 are not obvious in view of the teachings of Mansour and Schweickart, Applicants respectfully request withdrawal of the rejection under 35 U.S.C. § 103 (a).

It is believed that the claims are currently in condition for allowance, and notice to that effect is respectfully requested. The Commissioner is hereby authorized to charge any deficiency or credit any overpayment to Deposit Account No. 50-1271 under Order No. R-611.

Respectfully submitted,

Date: Aug 12, 2002


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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/815,937	03/22/2001	Keith D. Allen	R-611	1801

26619 7590 03/11/2002

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EXAMINER

QIAN, CELINE X

ART UNIT

PAPER NUMBER

1636

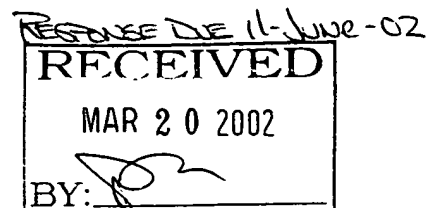
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Office Action Summary

Application No.

09/815,937

Applicant(s)

ALLEN ET AL.

Examiner

Celine Qian

Art Unit

1636

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 04 February 2002.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 1-38 is/are pending in the application.
- 4a) Of the above claim(s) 11-16 and 25-38 is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 1-10 and 17-24 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
- 11) ☐ The proposed drawing correction filed on _____ is: a) ☐ approved b) ☐ disapproved by the Examiner.
If approved, corrected drawings are required in reply to this Office action.
- 12) ☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

- 13) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
a) ☐ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
2. ☐ Certified copies of the priority documents have been received in Application No. _____.
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
* See the attached detailed Office action for a list of the certified copies not received.
- 14) ☒ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).
a) ☐ The translation of the foreign language provisional application has been received.
- 15) ☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

- 1) ☒ Notice of References Cited (PTO-892) 4) ☐ Interview Summary (PTO-413) Paper No(s). _____
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948) 5) ☐ Notice of Informal Patent Application (PTO-152)
- 3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) g. 6) ☐ Other: _____

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DETAILED ACTION

Claims 1-38 are pending in the application.

Election/Restrictions

Applicant's election with traverse of Group I in Paper No. 11 is acknowledged. The traversal is on the ground(s) that the inventions in Groups I-VI are related and thus a search can be made without serious burden on the Examiner. Specifically, Applicants argue that the methods of Groups II and III, II and V, III and V share a common starting material, the transgenic knockout mouse, therefore, the inventions are related. This is not found persuasive because of the following reasons. While the Examiner agrees with Applicants' arguments with regard to Groups II and III, II and V, III and V that they share a common starting material, the transgenic knockout mouse, however, is not the only starting material in these methods. The agents being tested are also part of starting material and they are different for each group. For instance, an agent that modulates the expression of a gene is different from an agent that modulates the function of said gene. Therefore, the inventions of Groups I-VI are patentably distinct for the same reasons set forth in the prior office action mailed on 12/3/01. The Examiner agrees that the inventions are related, however, the inventions are patentably distinct, and the search as required for the different groups are not co-extensive. Therefore, a search of all the groups will be a serious burden.

The requirement is still deemed proper and is therefore made FINAL.

Accordingly, claims 11-16 and 25-38 are withdrawn from consideration as being drawn to non-elected inventions. Claims 1-10 and 17-24 are currently under examination.



Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claim 8 is rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a lymphoid specific GPCR gene knockout mouse exhibiting the phenotype of lymphocyte infiltration, does not reasonably provide enablement for any other lymphoid specific GPCR gene knockout non-human animals without said phenotype. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make the invention commensurate in scope with these claims.

The nature of the invention is a lymphoid specific GPCR gene knockout non human animal. The specification discloses the generation of a mouse having lymphoid specific GPCR gene disrupted by homologous recombination in mouse ES cells and said mouse exhibit the phenotype of lymphocyte infiltration in lung, pancreas and liver (see page 62).

Since homologous recombination is required for gene targeting method as employed by the specification, embryonic stem cell technology must be available to carry out the method. At the time of the invention, no embryonic stem cell other than mouse was isolated as indicated by the art (Osterrieder & Wolf, Rev. sci. tech. Off. int. Epiz., vol. 17, no.1, 351-364, 1998). The guidance of specification is limited and does not provide a method as to obtain ES cells from other animals. The specification only teaches generation of a lymphoid specific GPCR gene knockout mouse by using ES cells containing disrupted lymphoid specific GPCR gene achieved by homologous recombination. The specification does not teach the generation of ES cells of

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other non-human animals. Further the specification does not teach the generation of a lymphoid specific GPCR gene knockout non human animal by other methods. Lack of guidance from specification, one skilled in the art would turn to prior art for guidance to make a lymphoid specific GPCR gene knockout non-human animal other than mouse. However, the prior art does not teach gene targeting methods other than homologous recombination. The art does not teach how to generate embryonic stem cells from non human animals other than mouse. Therefore, one skilled in the art would have to engage in undue amount of experimentation to make the claimed invention, a lymphoid specific GPCR gene knockout non-human animal.

Claim 8 does not recite any particular phenotype for a transgenic non-human animal comprising a disruption in a lymphoid specific GPCR gene. However, the phenotype exhibited by the lymphoid specific GPCR gene knockout non-human animal as a consequence of gene knockout, as disclosed in the specification, is required to enable the use of the non-human animal to identify agents that ameliorates lymphocytes infiltration, for example. The specification does not teach how to use the non-human animals lacking this phenotype. Therefore, one skilled in the art would have to engage in undue amount of experimentation to use the claimed invention.

Claims 17-23 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a homozygous lymphoid-specific GPCR knockout mouse lacks production of functional GPCR protein, does not reasonably provide enablement for a heterozygous lymphoid-specific GPCR knockout mouse or a lymphoid-specific gene disrupted mouse. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

The nature of the invention is a transgenic mouse comprising a disruption in a lymphoid-specific GPCR gene and exhibits phenotype comprising lymphocytes infiltration in lung, pancreas, stomach and liver; and a method of making said transgenic mouse. The specification discloses a method for generating said mouse by homologous recombination using a lymphoid specific GPCR targeting construct (see page 57-62, examples 1-4). The specification further discloses that the homozygous knockout mice exhibit the phenotype comprising lymphocytes infiltration in lung, pancreas, stomach and liver (see page 62, lines 8-24).

When considering the predictability of this invention, one has to remember that many of the phenotypes examined in transgenic knockout models are influenced by the genetic background in which they are studied and the effect of allelic variation and the interaction between the allelic variants (pg.1425, col.1 1st paragraph, Sigmund, C.D. 2000. *Arterioscler Thromb Vasc Biol.*20:1425-1429). The specification discloses the phenotype of a homozygous lymphoid-specific GPCR knockout mouse. Claims 17-20, 23 and 24 encompass heterozygotes, but since heterozygotes have one functional allele, the heterozygotes would not be expected to have the same phenotype as the homozygotes. Thus, the phenotype of a lymphoid-specific GPCR knockout mouse is unpredictable.

The specification discloses that the word "disruption" comprises alter or replace a promoter, enhancer, or splice site of a target gene, and can alter the normal gene product by inhibiting its production partially or completely or by enhancing the normal product's activity (see page 9, lines 19-26). However, it is not known in the prior art that such "disruption," would produce the phenotype as disclosed by the specification. The specification only discloses a mouse with two alleles of lymphoid-specific GPCR disrupted by inserting a selection marker that

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exhibits the phenotype comprising lymphocytes infiltration. Thus, the phenotype of a transgenic mouse comprising a "disruption," as defined by the specification, in a lymphoid-specific GPCR gene is unpredictable. Thus, the specification, in the instant case, is not enabling for transgenic knockout animals that exhibit no phenotype or that exhibit transgene-dependent phenotypes other than that disclosed in the instant specification. One skilled in the art would have to engage in undue amount of experimentation to make and use the invention commensurate in scope with these claims.

The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 1-4, 9, 10, 17, 18, 20-24 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Regarding claims 1-4 and 10, it is unclear how the target construct is arranged. In other words, is the first polynucleotide adjacent to the second polynucleotide or there is a selectable marker in between? In addition, it is also unclear whether the first and second polynucleotide is a contiguous sequence of the target gene or just portions of the target gene. As such, the metes and bounds of the claim cannot be established.

Regarding claims 9 and 24, the word "derived" renders the claim indefinite because the nature and number of derivative processes is unknown.

Regarding claims 17, 18 and 20-24, the term "cellular infiltration" renders the claims indefinite because the nature of the cell and the location the cell infiltrates are unknown.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 1-8 and 10 are rejected under 35 U.S.C. 103(a) as being unpatentable over Mansour et al (1988, Nature, vol. 336, No. 24, 348-352), in view of Schweickart et al. (1994, Genomics vol. 23, 643-650).

The claims are drawn to a lymphoid-specific GPCR gene-targeting construct and a method of making said construct. The claims are further drawn to a cell comprising a disruption in a lymphoid-specific gene, and a method of producing a transgenic mouse comprising a disruption in a lymphoid-specific GPCR gene by homologous recombination using the target construct.

Mansour et al. teach a strategy for targeted disruption of the *hprt* and proto-oncogene *int-2* in mice embryonic stem cells and subsequent generation of knockout mice. Their teaching

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addresses the previous technical difficulty of obtaining embryonic stem cell carrying non-selectable, targeted gene mutation at loci of interest, and therefore provides a model which can be used to produce homozygous mutation of any gene, regardless of its function, if a cloned fragment of the gene is available (see page 348, second paragraph, line 1-3, third paragraph, line 1-5, and page 352, fourth paragraph, line 1-3). Mansour et al. further teach the generation of two targeting constructs, pRV9.1/TK and pINT-2-N/TK, each contains two sequences from hprt and int-2 respectively, and a neo selection marker in between the two sequences (see page 350, figure 3). However, Mansour et al. do not teach how to make a lymphoid-specific GPCR gene target construct and knockout mouse.

Schweickart et al. teach the cloning of human and mouse lymphoid-specific GPCR gene EBI1. They provide the cloned coding sequence for lymphoid-specific GPCR gene (see page 645, figure 1). Schweickart et al. also teach that EBI1 is highly homologous to several members of the leukocyte chemotactic peptide receptor family and its expression is specific to lymphoid organs (see page 648, 1st col., 3rd paragraph). Schweickart et al. further teach that this receptor play a role in lymphocyte growth, differentiation, activation, leukocyte trafficking, and in the extravasation of blood cells into sites of inflammation (see page 648, 1st col., last line, 2nd col., 4th paragraph, line 1-2).

It would have been obvious to one in the ordinary art to make a lymphoid-specific GPCR knockout mouse. The ordinary artisan would have been motivated to knockout the function of lymphoid-specific GPCR gene in a mouse to study the role this gene plays in lymphocyte growth and regulation (see page 648, 1st col., last line, 2nd col., 4th paragraph, line 1-2). The ordinary artisan would have had reasonable expectation of success because of the teachings of Mansour et

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al., who teach a general method of targeted gene disruption in mice based on homologous recombination using a cloned fragment of a desired gene, and Schweickart et al., who teach the coding sequence of the mouse lymphoid-specific GPCR gene, and also teach the importance of this gene in regulating lymphocyte growth, differentiation, activation and migration. Therefore, the invention would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Celine X Qian whose telephone number is 703-306-0283. The examiner can normally be reached on 9:00-5:30 M-F.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Dr. Remy Yucel can be reached on 703-305-1998. The fax phone numbers for the organization where this application or proceeding is assigned are 703-305-3014 for regular communications and 703-305-3014 for After Final communications.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the receptionist whose telephone number is 703-308-0196.

Celine Qian, Ph.D.
March 11, 2002


REMY YUCEL, PH.D
SUPERVISORY PATENT EXAMINER
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Notice of References Cited

Application/Control No.

09/815,937

Applicant(s)/Patent Under
Reexamination
ALLEN, KEITH D. ET AL

Examiner

Celine Qian

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U.S. PATENT DOCUMENTS

*		Document Number Country Code-Number-Kind Code	Date MM-YYYY	Name	Classification
	A	US-			
	B	US-			
	C	US-			
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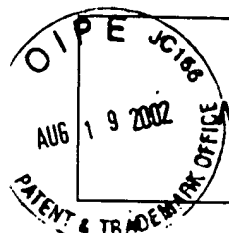
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	S					
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NON-PATENT DOCUMENTS

*		Include as applicable: Author, Title Date, Publisher, Edition or Volume, Pertinent Pages)
	U	Osterrieder et al., Lessons from gene knockouts, 1998, REV. SCI. TECH. OFF. INT. EPIZ., Vol. 17, pp. 351-364
	V	Mansour et al., Disruption of the proto-oncogene int-2 in mouse embryo-derived stem cells: a general strategy for targeting mutations to non-selectable genes, 1988, NATURE, Vol. 336, pp. 348-352
	W	Sigmund, Viewpoint: Are studies in genetically altered mice out of control?, 2000, ARTERIOSCLER THROMB. VASC. BIOL., Vol. 20, pp. 1425-1429
	X	Wall, Transgenic livestock: Progress and prospects for the future, 1996, THERIOGENOLOGY, Vol. 45, pp. 57-68

*A copy of this reference is not being furnished with this Office action. (See MPEP § 707.05(a).)
Dates in MM-YYYY format are publication dates. Classifications may be US or foreign.



Notice of References Cited

Application/Control No.

09/815,937

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*		Include as applicable: Author, Title Date, Publisher, Edition or Volume, Pertinent Pages)
*	U	Schweickart et al., Cloning of human and mouse EB11, a lymphoid-specific G-protein-coupled receptor encoded on human chromosome 17q12-q21.2
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*A copy of this reference is not being furnished with this Office action. (See MPEP § 707.05(a).)
Dates in MM-YYYY format are publication dates. Classifications may be US or foreign.